tests. The pilots were divided into 2 groups according to the frequency of VPBs detected on 24-hour Holter monitoring: Group 1: Pilots with rare VPBs, Group 2: Pilots with frequent VPBs. Data was recorded and analyzed automatically by the software of the Holter device. Statistical analyses were done by using SPSS-15 software.
Results: There were no differences in terms of their age, minimum and maximum heart rates, supraventricular extrasystoles but the average heart rate which was found to be lower in group 2. SDNN Indeks, RMSSD and PNN50 parameters were significantly higher in Grup-2. Data of both groups are shown in Table-1.
Conclusion: Exposure to both acceleration forces (G) and anti-G protective maneuvers cause changes in cardiac preload and afterload. Although the heart beats harder and faster to copy with these changes by sympathovagal interaction, chronic +Gz exposure has no effect on cardiac dimensions and structure. But it may have some effects on the SA node and electrical conduction system of the heart. We found lower heart rates, higher SDNN Indeks, RMSSD and PNN50 parameters suggestive of higher risk for incapacitation tendency were found in pilots with frequent VPBs compared to the other group.

Table 1. HRV Parameters of Pilots with VPBs

Parameters	Group 1 N= 21	Group 2 N= 32	P (<0.05)
Age	36.05±5.37	33.00±5.93	0.071
VE	356.57±263.27	3887.41±2686.82	0.000
SDNN 24 hour (ms)	153.62±29.75	158.56±35.59	0.778
SDANN index (ms)	145.14±40.15	145.63±34.30	0.792
SDNN index (ms)	60.76±9.73	69.09±15.69	0.028
RMSSD (ms)	31.29±6.15	39.22±14.67	0.043
PNN50 (%)	8.43±4.10	15.06±10.08	0.021
SP24h (ms2)	3872.11±1233.73	4783.13±1977.60	0.127
VLF (ms2)	2544.56±946.26	3182.79±1462.19	0.122
LF (ms2)	1047.04±362.59	1247.31±483.75	0.098
HF (ms2)	260.71±131.75	330.06±216.45	0.283
Min SPH (ms2)	1400.85±852.25	1908.68±1150.16	0.091
Max SPH (ms2)	9017.29±4589.98	10554.05±5849.72	0.252
SVE	978.14±2112.52	996.69±1855.49	0.519
Min HR	44.19±4.46	41.75±5.41	0.097
Max HR	147.43±25.40	139.34±25.31	0.167
Average HR	78.10±5.44	74.19±8.14	0.039

HR = heart rate, VE = ventricular extrasystole, SDNN = standard deviations of all NN intervals, SDANN = the standard deviation of the average NN intervals calculated over 5 minutes periods, RMSSD = the square root of the mean of the sum of the squares of differences between adjacent NN intervals, PNN50 = the number of pairs of adjacent NN intervals differing by more than 50 ms divided by the total number of all NN intervals, SP24h = 24 hour spectral power, SVE = supraventricular extrasystole, VLF = very low frequency range power (0.003-0.04 Hz), LF = low frequency range power (0.04-0.15 Hz), HF = high frequency range power (0.15-0.40 Hz)

PP-164

Angiotensin-Converting Enzyme Insertion/Deletion (I/D) Polymorphism Associated with Atrial Fibrilation in Turkish Population

Atilla İçli[1], Nilgün Erten[2], Recep Sütçü[3], Fatih Aksoy[4], Akif Arslan[5], Habil Yücel[6], Mehmet Koray Adalı[2]
[1]Department of Cardiology, Ahi Evran University Training and Research Hospital, Kırsehir, [2]Department of Neurology, Giresun University, Giresun, [3]Department of Biochemistry, Katip Celebi University, İzmir, [4]Department of Cardiology, Suleyman Demirel University, Isparta, [5]Department of Cardiology, Aksaray State Hospital, Aksaray, [6]Department of Cardiology, Isparta State Hospital, Isparta

Background: Atrial fibrillation (AF) is the most commonly observed arrhythmia in clinical practice and associated with increased cardiovascular morbidity and mortality. The renin-angiotensin system may play a role in the pathogenesis of atrial fibrillation. Increased angiotensin-converting enzyme (ACE) expression in the atrial tissue of patients with AF has suggested the involvement of the RAS in AF. Some initial studies indicated an association between an angiotensin-converting enzyme insertion/deletion (ACE I/D) polymorphism and AF, however, the results have been inconsistent. We aimed to investigate relationship between AF and polymorphism of ACE I/D in Turkish patients.
Methods: Sixty eight patients with permanent AF and 65 patients with no documented episode of AF matched for age, race and sex. Because ethnic differences have been reported for ACE I/D polymorphism. The ACE I/D gene polymorphism was identified by polymerase chain reaction (PCR) method. The I/D polymorphism of the ACE gene was assessed by detecting the presence (allele I, insertion) or absence (allele D, deletion) of a 287-bp sequence in the intron 16 of the ACE gene in the chromosome 17. Distribution of the ACE I/D gene polymorphism alles (allele I, insertion, allele D, deletion) genotypes (DD, ID and II) were determined in study population. Demographic characteristics and risk factors for atrial fibrillation were evaluated in the study groups.
Results: There was no significant difference with respect to age and gender between groups. Genotype and allel distribution of AF(+) and AF(-) groups shown in the table. The frequency of II genotype of ACE I/D polymorphism was significantly lower in patients with AF(+) group compared with AF(+) group (13 (19.1%) vs 25 (38.5%), p=0.014). The frequency of DD genotype homozygous genotype was significantly higher in AF(+) group than AF(-) (32 (47.1%) vs 19 (29.2%), p=0.035). Between the two groups were compared according to the dominant genetic model (ID+DD vs. II), The number of patients carrying at least one D mutant allele (ID+DD) was significantly higher in AF(+) group than AF (-) group (55 (80.9%) vs 40 (61.5%), p=0.014). With respect to allelic distribution (I vs D, additive model), the frequency of the D allele was significantly higher in AF patients. (89 (65.4%) vs 60 (46.1%), p=0.021).
Conclusions: In this study, our data suggest that the ACE I/D gene polymorphisms may be assassed as a risk factor in the occurance of AF. However, further large-sized studies are required for determining relationship between ACE I/D gene polymorphisms and AF.

Angiotensin-converting enzyme insertion/deletion gene polymorphism genotype and allel frequencies

	AF (+) patients (n:68)		AF (-) patients (n:65)		P
	n:	%	n:	%	
II genotype	13	19.1	25	38.5	0.014
ID genotype	23	33.8	21	32.3	0.853
DD genotype	32	47.1	19	29.2	0.035
ID+ DD genotypes (Dominant genetic model)	55	80.9	40	61.5	0.014
D allel	89	65.4	60	46.1	0.021

PP-165

Baseline ECG Parameters in Turkish Population: Results from the HAPPY Study

Burak Hünük[1], Özgür Çağaç[2], Okan Erdoğan[2], Alper Kepez[2], Bülent Mutlu[2], Muzaffer Değertekin[3], Çetin Erol[4]
[1]Maltepe C.I.K. State Hospital, Department of Cardiology, Istanbul, [2]Marmara University, Faculty of Medicine, Department of Cardiology, Istanbul, [3]Yeditepe University, Faculty of Medicine, Department of Cardiology, Istanbul, [4]Ankara University, Faculty of Medicine, Department of Cardiology, Ankara

Purpose: Traditionally, ECG reference ranges are derived from studies carried in different populations or trial data. However, ECG values differ between populations, gender and age groups. Hence, our aim was to determine the baseline ECG parameters in Turkish population.
Methods: ECGs were obtained from the HAPPY (Heart Failure Prevalence and Predictors in Turkey) study involving randomly selected 4650 subjects ≥35 years from all geographical regions of Turkey. After the exclusion of subjects with missing ECG or data, antiarrhythmic use and any "abnormal" ECG findings (bundle branch blocks, pre-excitations, atrial fibrillation, hypertrophies); 3016 subjects ([mean±SD] age, 51±11, [range]35-100 years) were enrolled in the study (female n [overall %]:1765 [58,5%]). ECGs were interpreted manually by two experienced cardiologists for baseline intervals.
Results: The baseline ECG parameters in each age group are shown in Table-1. Overall, women had significantly higher resting heart rates, wider QT/rate corrected QT (QTc-Bazett's formula), narrower PR intervals and QRS durations (p<0,001).
Conclusions: ECG baseline parameters in Turkish population resembles to other study results conducted in Caucasian populations. In subjects ≥65 years old, distinct features found in young female population diminished and both sexes demonstrated similar ECG parameters. Further models are needed in clinical practice to reliably classify any surface ECG of different race/age/gender/rate as "abnormal".

Table 1

		Mean ± SEM (1st - 99th percentiles)							
Age (years)	Gender (n)	QTc (ms)		PR (ms)		QRS (ms)		Rate (beats/ min)	
35-54	F(1133)	406,4 ± 0,48 (368-446)	p< 0,001	151,3± 0,58 (112-203)	p< 0,001	87,0± 0,25 (68-112)	p< 0,001	74.8± 0,34 (52-107)	p< 0,001
	M(733)	402,2± 0,64 (362-442)		155,7± 0,77 (114-223)		93,4± 0,36 (72-116)		71,8± 0,41 (49-102)	
55-64	F (352)	407,6± 0,91 (368-450)	p= 0,008	155,2± 1,14 (110-216)	p= 0,033	87,5± 0,46 (70-110)	p= 0,010	75,2± 0,60 (53-107)	p= 0,013
	M(306)	404,0± 0,99 (371-447)		158,8± 1,27 (112-231)		89,9± 0,55 (66-116)		72,9± 0,71 (50-110)	
≥65	F (280)	409,3± 1,13 (356-447)	p= 0,440	158,9± 1,45 (119-236)	p= 0,228	85,3± 0,55 (65-110)	P< 0,001	73,2± 0,68 (50-112)	p= 0,324
	M(212)	408,0± 1,261 (368-452)		161,5± 1,58 (112-238)		90,7± 0,68 (68-115)		72,2± 0,84 (51-103)	
	Overall (3016)	405,7± 0,31 (368-446)		155,0± 0,38 (112-218)		89,1± 0,17 (68-114)		73,59± 0,21 (51-106)	

SEM: Standart Eror of Mean, p: intra age group significance of difference between gender, F: Female subjects, M: Male subjects

PP-166

The Impact of Anemia on QT Interval: A Population Based Study

Burak Hünük[1], Özgür Çağaç[2], Okan Erdoğan[2], Alper Kepez[2], Bülent Mutlu[2], Muzaffer Değertekin[3], Çetin Erol[4]
[1]Maltepe C.I.K. State Hospital, Department of Cardiology, Istanbul, [2]Marmara University, Faculty of Medicine, Department of Cardiology, Istanbul, [3]Yeditepe University, Faculty of Medicine, Department of Cardiology, Istanbul, [4]Ankara University, Faculty of Medicine, Department of Cardiology, Ankara

Purpose: The prolongation of QT interval/rate corrected QT interval (QT/QTc) is associated with cardiovascular (CV) mortality and morbidity. Anemia is an independent risk factor for CV outcomes. Effects of age and gender on QT interval have been well documented. However, the impact of anemia on QT remains to be elucidated.
Methods: ECGs were obtained from the HAPPY (Heart Failure Prevalence and Predictors in Turkey) study including randomly selected 4650 subjects ≥35 years with laboratory and clinical data from Turkey. After the exclusion of subjects with missing data, ECG abnormalities effecting QT, anti arrhythmic use or established coronary artery disease; 3374 subjects ([mean±SD]age, 51±11, [range]35-100 years) were enrolled in the study (female n [overall%]:1943 [57,6%]). Anemia was defined according to the WHO criteria (hemoglobin[Hgb] <13 g/dl in men, <12 g/dl in women). Long QTc was defined if interval was >440 ms for men and >460 ms for women. ECGs were interpreted by two experienced cardiologists for baseline ECG parameters and automated QTc analysis (Bazett's formula) values.
Results: Long QTc was detected in 1,8% of the population. Three hundred eighty one women (19,6% of females) and 150 men (10,5% of males) were anemic. The incidence of anemia was higher in long QTc group vs normal QTc group (31,6% vs 16,2%, p=0,003). QTc values were significantly longer in patients with anemia (Data±SEM, 408±0,8 vs 406±0,3, p=0,02). Hgb levels were negatively correlated with QTc in univariate analyses (r=-0,101, p<0,001). In multivariate regression analyses, Hgb was found as an independent predictor of QTc durations after adjusting for age, gender and BMI (Table-1).
Conclusions: Anemia seems to prolong QT interval in subjects without a manifest heart disease. Our findings might be attributed to a probable subendocardial ischemia caused by a demand/supply mismatch in the coronary physiology of anemic subjects.

Table 1

Parameters	Univariate		Multivariate		
	r	p	β	p	Confidence Interval (95%)
Age (years)	0,100	< 0,001	0,107	< 0,001	(0,111 - 0,216)
Gender	0,092	< 0,001	0,087	0,001	(0,970 - 3,937)
BMI (kg/m2)	0,029	0,040	0,003	0,850	(-0,116 - 0,141)
Hgb (g/dl)	-0,101	< 0,001	-0,059	0,005	(-1,129 - -0,202)

Multivariate regression model for QTc

PP-167

Impact of Metabolic Factors on QT Interval: Results from the HAPPY Study

Burak Hünük[1], Özgür Çağaç[2], Okan Erdoğan[2], Alper Kepez[2], Bülent Mutlu[2], Muzaffer Değertekin[3], Çetin Erol[4]
[1]Maltepe C.I.K. State Hospital, Department of Cardiology, Istanbul, [2]Marmara University, Faculty of Medicine, Department of Cardiology, Istanbul, [3]Yeditepe University, Faculty of Medicine, Department of Cardiology, Istanbul, [4]Ankara University, Faculty of Medicine, Department of Cardiology, Ankara

Purpose: It is well known that the prolongation of QT interval is associated with cardiovascular mortality and morbidity. The effects of age and gender on QT/rate corrected QT interval (QTc) have previously been investigated. However, metabolic factors on QT interval is not well established. Our aim was to investigate the effects of body mass index (BMI), metabolic syndrome (MS), diabetes mellitus (DM), glomerular filtration rate (GFR), hypertension (HT) and mean arterial pressure (MAP) on QTc.
Methods: ECGs were obtained from the HAPPY (Heart Failure Prevalence and Predictors in Turkey) study which included randomly selected 4650 subjects ≥35 years with laboratory and clinical data from Turkey. After the exclusion of subjects with missing ECG or data, ECG abnormalities effecting QTc, anti arrhythmic use or established coronary artery disease; 2984 subjects ([mean±SD]age, 50±11, [range] 35-80 years) were enrolled in the study (female n [overall%]:1723 [58,6%]). MS was defined according to the revised third National Cholesterol Education Program Adult Treatment Panel. GFRs were calculated by Cockroft-Gault formula and grouped into high (≥60 ml/min) and low (<60 ml/min).
Results: QTc was significantly longer in MS (meanQTc±SEM; 406,6±0,5 vs 404,9±0,4 p=0,005), HT (406,5±0,5 vs 405,0±0,4, p=0,014), low GFR (409±1,2 vs 405,6±0,3, p=0,007) and high BMI group (407,1±0,6 vs 405±0,4, p=0,002). There was a significant negative correlation with GFR and QTc (r:-0,047, p=0,007). Multivariate regression model for QTc is shown in table-1.
Conclusions: After adjusting for MAP and GFR; age and BMI revealed as independent predictors of QTc in adult Turkish population. This finding might be attributed to a probable sub-clinical cardiac hypertrophy or chamber enlargement in high BMI.

Table 1

Parameters	Univariate		Multivariate		
	r	p	β	p	Confidence Interval (95%)
Age (years)	0,096	< 0,001	0,073	0,001	(0,050-0,178)
BMI (kg/m2)	0,066	< 0,001	0,067	0,001	(0,1-0,4)
GFR (ml/min)	-0,047	0,007	0,008	0,697	(-0,035-0,053)
MAP (mmHg)	0,079	0,001	0,041	0,058	(-0,002-0,106)

Multiple regression analysis of variables predictive of QTc